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Mediastinal Emphysema During Diabetic Ketoacidosis

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DIRECT COMPLICATIONS of diabetic ketoacidosis, such as hypovolemic renal failure and extremes of serum potassium, are well known to practicing physicians. In addition to the anticipated metabolic derangements, concomitant problems such as gastric or intestinal atony, seizures, cerebral edema, acute flaccid quadriplegia or severe muscular weakness, polyneuropathy or fulminant infections are occasionally encountered. The following brief case report illustrates yet another apparently associated complication of diabetic ketoacidosis, mediastinal emphysema.

Case Report

A 12-year-old Caucasian boy was admitted to a small private hospital on 20 October 1968 with

a history of loss of 15 pounds in weight over a period of three weeks and a two-day period of intense thirst, irritability, abdominal pain of a diffuse nature and vomiting. Until then the patient had been in excellent health. He was not known to be diabetic and family history was negative in this regard. He was an only child.

On physical examination he appeared anxious and was hyperventilating. Temperature was 35.6° C (96° F), pulse of 160 per minute, blood pressure of 120/60 mm of mercury and respirations 40 per minute and deep. The eyeballs were sunken in the orbits and mucous membranes were dry. The neck was supple without adenopathy and there was no subcutaneous emphysema. With the patient supine the neck veins were not distended. The chest was clear. Cardiac tones were heard well and the left heart border was within the mid-clavicular line. A loud crunching type of sound was heard along the left sternal border synchronous with systole. The abdomen was flat, bowel sounds were absent, and there was moderate guarding with no rebound tenderness. All peripheral pulses were intact. The skin was warm and dry. The neurological examination was unremarkable except for pronounced agitation and at times confusion. The patient's breath had a strong acetone odor.

Results of laboratory studies at the time of admission were: hemoglobin 15.2 grams per 100 ml; hematocrit, 45 percent; white blood cell count 20,000 per cu mm with a shift to the left; blood urea nitrogen 21.8 mg per 100 ml; blood glucose 575 mg per 100 ml; blood acetone positive at a 1:8 dilution; serum sodium 135 mEq per liter; serum potassium 5.2 mEq and serum bicarbonate 6.3 mEq per liter; blood pH 6.98; blood pCO₂ 13.5 mm of mercury; urine had a specific gravity of 1.028 with 4+ glucose and 4+ acetone. A throat culture later grew *Staphylococcus aureus* coagulase-positive, but two blood cultures drawn on admission remained negative.

Initial treatment consisted of 40 units of regular insulin and 44 mEq of sodium bicarbonate intravenously. During the first eight hours of therapy, the patient received a total of 4300 ml of fluids, either Ringer's lactate or half-normal saline solution, containing 120 mEq of potassium chloride. A total of 270 units of regular insulin was administered at intervals during this period.

Additional management included nasogastric intubation with continuous gastric suction, seda-

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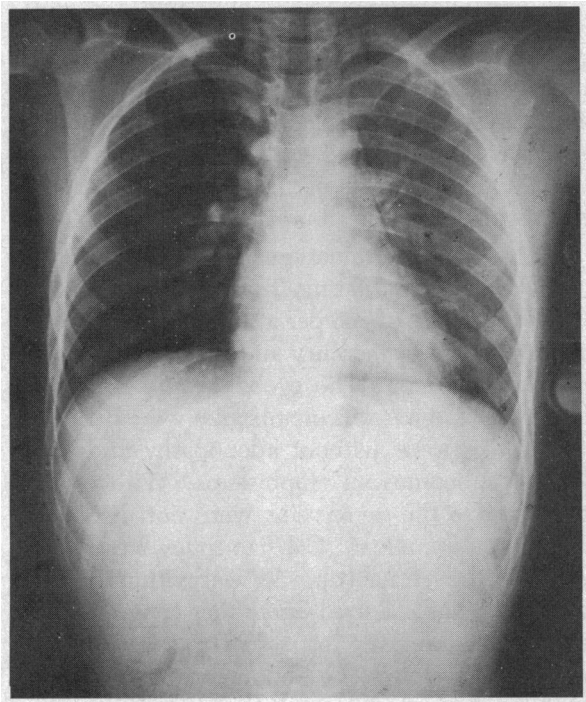


Figure 1.—X-ray film taken shortly after admission shows air in the mediastinum and pericardial sac.

tion with frequent small doses of intravenous chlordiazepoxide, and antibiotics.

An x-ray film of the chest taken shortly after admission revealed large amounts of air in the mediastinum and pericardial sac (Figure 1). There was no evidence of pulmonary infiltration or pneumothorax. A plain film of the abdomen showed no abnormality; in particular there was no free air beneath the diaphragm or retroperitoneal air lines.

Because of an abrupt temperature rise to 38.2° C (100.8° F) and a suspicion of infection with a gas-forming organism, the patient was treated with large doses of penicillin and kanamycin. The abdominal pain abated over the next 12 hours. An x-ray film the next day showed reduction of the mediastinal air (Figure 2). The following day the patient was afebrile. Complete restoration of blood chemical factors and electrolytes took 72 hours.

Discussion

The occurrence of mediastinal emphysema (pneumomediastinum) during the course of diabetic ketoacidosis has not been appreciated until the past several years.¹⁻⁴ We were unaware of this at the time the patient was being managed,

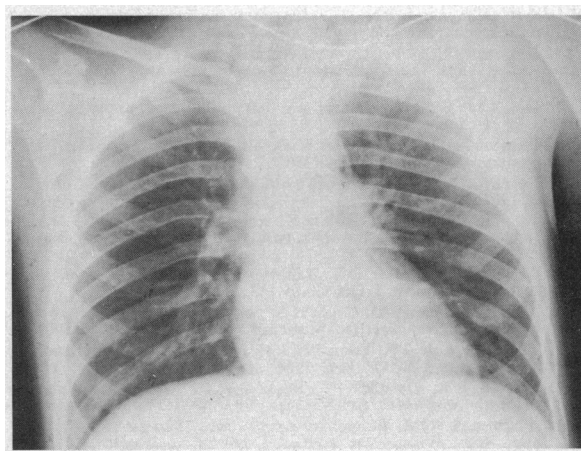


Figure 2.—X-ray film the day after admission and following treatment with penicillin and kenamycin shows reduction of mediastinal air.

and the presence of air in the mediastinum reminded us of a recent report of diabetic ketoacidosis with pneumointestinalis secondary to bacterial invasion of the colon.⁵ The rapid recovery of the patient, however, eliminated infection due to a gas-forming organism as the cause for the mediastinal emphysema.

The underlying causes of mediastinal emphysema are numerous and varied. The more common conditions associated with or leading to its appearance include mediastinal trauma, perforation of an abdominal viscus with leakage of air through the diaphragmatic orifices, atelectasis, and parturition. Conditions resulting in expiratory resistance, such as bronchial asthma, inhalation of foreign bodies, positive pressure therapy, and a variety of respiratory infectious diseases are also prime culprits.⁶⁻⁸

The pathogenetic mechanisms involved in the production of mediastinal air, according to the Macklins, begin with a pressure differential across the walls of the marginal alveoli leading to their rupture. Pulmonary interstitial emphysema ensues, air then dissects proximally by way of the connective tissue sheaths of the pulmonary blood vessels.⁹

Concerning the possible relationship of diabetic ketoacidosis to mediastinal emphysema, several observers have suggested that the retching and vomiting, so common in the ketotic patient, are responsible for the abrupt distention and tearing

of alveolar septae.³ Others implicate the Kussmaul-type breathing itself.² It would be of interest to learn if this complication occurs also with other forms of metabolic acidosis with deep hyperventilation, such as acute salicylism. In any event it is apparent that an association between these two entities does exist.

Treatment directed toward the mediastinal emphysema *per se* is usually unnecessary. If tension occurs, incision above the suprasternal notch to allow air to escape or simple percutaneous puncture of the resultant supraclavicular subcutaneous emphysema suffices, although some authorities recommend mediastinotomy.⁸

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A HOMESPUN GAUGE OF NEUROLOGIC DEFICIT

"If you were on a desert island and had no laboratory tests or anything else to use in examining young children for neurologic deficits, you could use a tape measure. I think that this is the single most under used and most valuable technique for evaluating deficit in children. . . .

"There are two ways you can use the information from a tape measure. First you can plot the head circumference on a chart. A study from Denver, which puts together all of the available data in the literature, shows that there is very little variation from one nationality or race group to another in terms of the normal head size. If you find, for example, that the head circumference deviates from the mean as much as two standard deviations, I think you've got important diagnostic information, especially in an infant who is behind developmentally, or experiencing seizures, or in whom you suspect central nervous system difficulties.

"An alternative way of using this information, one that I use regularly, is to simply obtain a normal head circumference measurement for boys and girls and put it in your wallet. . . . What we did in determining intracranial volume was to take a set of x-ray measurements and compare them with head circumference measurements; the degree of correlation was .985. That's why I think you really don't have to get fancy and you don't need the radiologist's evaluation of head size if you use the tape measure."

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